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Review article

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Effects of microplastics and nanoplastics on the kidney and cardiovascular system

Yu-Hsuan Lee[1,9], Cai-Mei Zheng[2,3,4], Ying-Jan Wang[5], Yung-Li Wang[6,7,9] & Hui-Wen Chiu[4,6,8]

Abstract

Microplastics and nanoplastics are ubiquitous environmental pollutants that contaminate air, food and water supplies, resulting in widespread human exposure and potential health risks. Varying concentrations of particulate plastics have been identified in human tissues and body fluids, including the heart, kidney, liver, brain, blood and urine. Studies in animal models and human cells have reported that particulate plastics can induce oxidative stress, cell death and inflammation as well as disrupt metabolism and immune function. They have also been shown to have toxic effects on kidney and cardiovascular cells, which are exacerbated by the presence of other environmental contaminants such as heavy metals. Patients with kidney failure might be at risk of increased exposure to particulate plastics during dialysis. Furthermore, clinical evidence suggests that particulate plastic exposure is a risk factor for cardiovascular disease. Approaches to mitigating such exposure include degradation via abiotic and biotic processes, improved waste management and water filtration approaches and use of alternative materials. Further research into the fate, toxicity and health consequences of particulate plastics is imperative to inform strategies to address this escalating environmental and health concern.

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[1]Department of Cosmeceutics, China Medical University, Taichung, Taiwan. [2]Division of Nephrology, Department of Internal Medicine, School of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan. [3]Division of Nephrology, Department of Internal Medicine, Shuang Ho Hospital, Taipei Medical University, New Taipei City, Taiwan. [4]TMU Research Center of Urology and Kidney, Taipei Medical University, Taipei, Taiwan. [5]Department of Environmental and Occupational Health, College of Medicine, National Cheng Kung University, Tainan, Taiwan. [6]Graduate Institute of Clinical Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan. [7]School of Dentistry, College of Oral Medicine, Taipei Medical University, Taipei, Taiwan. [8]Department of Medical Research, Shuang Ho Hospital, Taipei Medical University, New Taipei City, Taiwan. [9]These authors contributed equally: Yu-Hsuan Lee, Yung-Li Wang. e-mail: leu3@tmu.edu.tw

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Key points

- Particulate plastics are ubiquitous environmental pollutants that contaminate terrestrial and aquatic ecosystems, resulting in human exposure via ingestion, inhalation and skin contact.
- Following exposure, particulate plastics can enter the bloodstream and circulate throughout the body; they have been identified in organs including the heart, kidney, liver and brain.
- Studies in animal models and human cells have demonstrated that exposure to particulate plastics can induce oxidative stress and cytotoxic effects as well as disrupt energy homeostasis, metabolism and immune function.
- Evidence from in vitro experiments and animal studies suggests that particulate plastics have toxic effects on the kidney; patients receiving haemodialysis might be at risk of increased exposure to particulate plastics.
- Particulate plastics are potential risk factors for cardiovascular disease and have been shown to have adverse effects on cardiovascular cells in experimental studies, including induction of oxidative stress, inflammation, autophagy dysfunction and suppression of angiogenesis.
- Approaches to mitigating particulate plastic pollution include degradation via biotic and abiotic mechanisms; stricter regulations for plastic production, use and disposal; use of alternative materials; and public education efforts to encourage sustainable choices.

Introduction

Plastic degrades slowly and rapidly accumulates in the environment[1,2], resulting in the ubiquitous presence of particulate plastic pollution across terrestrial and aquatic ecosystems[3,4]. This pollution has garnered considerable public concern owing to its negative impact on the environment, harmful effects on living organisms and potential health risks. Importantly, the adverse effects of particulate plastics are likely to be increased in top predators, including humans, owing to biomagnification — the accumulation of plastics and associated toxins at higher levels of the food chain. As predators consume prey containing these contaminants, they experience amplified exposure, increasing health risks.

Mismanagement and inappropriate disposal of plastic waste is an important contributor to plastic pollution[5]. During the COVID-19 pandemic, the global production of plastic waste substantially increased to an estimated 1.6 million metric tonnes per day owing to the widespread use of personal protective equipment to mitigate viral transmission[6]. A 2024 study predicted that the annual amount of mismanaged plastic waste could nearly double to 121 million metric tons by 2050, with plastic-related greenhouse gas emissions rising by 37% to 3.35 gigatons CO_2 equivalent annually[7].

Plastic undergoes continuous fragmentation through 'ageing' processes, including exposure to ultraviolet radiation, fluctuations in temperature, mechanical abrasion and biotic processes[8]. Although no universal definitions exist, particulate plastics are generally classified based on their size as microplastics (MPs, <5 mm) and nanoplastics (NPs, <1 µm)[9,10]. However, the definitions of MPs and NPs vary between studies. The omnipresence of particulate plastics in consumer goods and the resulting environmental pollution results in widespread human exposure to these microscopic particles via the digestive system, skin and lungs[11]. After entering the blood, particulate plastics circulate throughout the body and can be found in various organs, including the heart, kidney, liver and brain[12–14].

The absorption of particulate plastics has diverse biological repercussions across multiple organ systems. For example, epidemiological studies have identified a high occurrence of interstitial lung disease among workers at nylon flock manufacturing plants in Canada and the USA, suggesting an occupational health risk associated with prolonged exposure to airborne synthetic fibres in industrial settings[15]. Ingestion of MPs has been associated with exacerbation of inflammatory bowel disease[16] and clinical evidence suggests that exposure to particulate plastics is a risk factor for cardiovascular disease[17]. Furthermore, an analysis of brain tissue samples obtained at autopsy found that those from patients with dementia showed increased accumulation of particulate plastics, particularly in cerebrovascular walls and immune cells[14]. Exposure to particulate plastics could potentially be increased among patients on long-term kidney replacement therapy owing to the possible contamination of haemodialysis fluid as well as the probable accumulation of particulate plastics owing to loss of kidney function[18].

In this Review, we discuss the impact of environmental exposure to particulate plastics on human health. We describe routes of exposure to MPs and NPs and potential mechanisms of toxicity in the kidney and cardiovascular systems, as well as the impact of contamination of haemodialysis fluids with particulate plastics and approaches to mitigating particulate plastic pollution.

Exposure to particulate plastics

MPs and NPs are pervasive environmental pollutants that contaminate food and water supplies and enter the human body through various pathways[19] (Fig. 1). Plastics can be ingested owing to contamination of food or drinking water or the use of plastic products, such as food packaging and utensils. Other routes of exposure include inhalation of air and dust that contains particulate plastics and skin contact with products such as clothing or cosmetics, or environments that are heavily laden with plastics[20]. A study that investigated exposure to MPs in Mumbai, India, estimated a total daily average intake of 2,012 ± 598 MPs per person via drinking water (382 ± 205 MPs), air (594 ± 269 MPs) and food (1,036 ± 493 MPs). The corresponding plastic intake ranged from 122.25 ± 177.38 mg to 202.80 ± 294.25 mg per day[21]. Other estimates suggest that adults may inhale 0.9×10^4 to 7.9×10^4 MPs and ingest up to 4.1×10^4 MPs from water and 5.1×10^3 MPs from salt per year[22].

A study that used a probabilistic model to simulate lifetime exposure to MPs based on food intake, inhalation, intestinal absorption, biliary excretion and plastic-associated chemical exposure estimated a median daily intake of 553 particles for children and 883 particles for adults, with substantial lifetime accumulation[23]. The researchers estimated that cumulative exposure to these daily intakes of MPs (1-10 µm) would result in irreversible accumulation within body tissues of approximately 8.32×10^3 particles by the age of 18, and up to 5.01×10^4 particles by the age of 70. They validated their model through stool analysis, which confirmed the presence of MPs. These findings highlight the importance of further investigating the potential long-term health effects associated with chronic MP exposure.

Absorption, distribution and excretion of particulate plastics

In the gut, particulate plastic absorption predominantly occurs via transcytosis across enterocytes; however, larger particles may be internalized through paracellular gaps (via persorption) or phagocytosed by immune cells[24]. In the lung, particulate plastics that are < 5 µm in size may reach the alveoli and be internalized via transcytosis, transported through the lymphatic system, phagocytosed by macrophages or cleared through mucociliary transport[24].

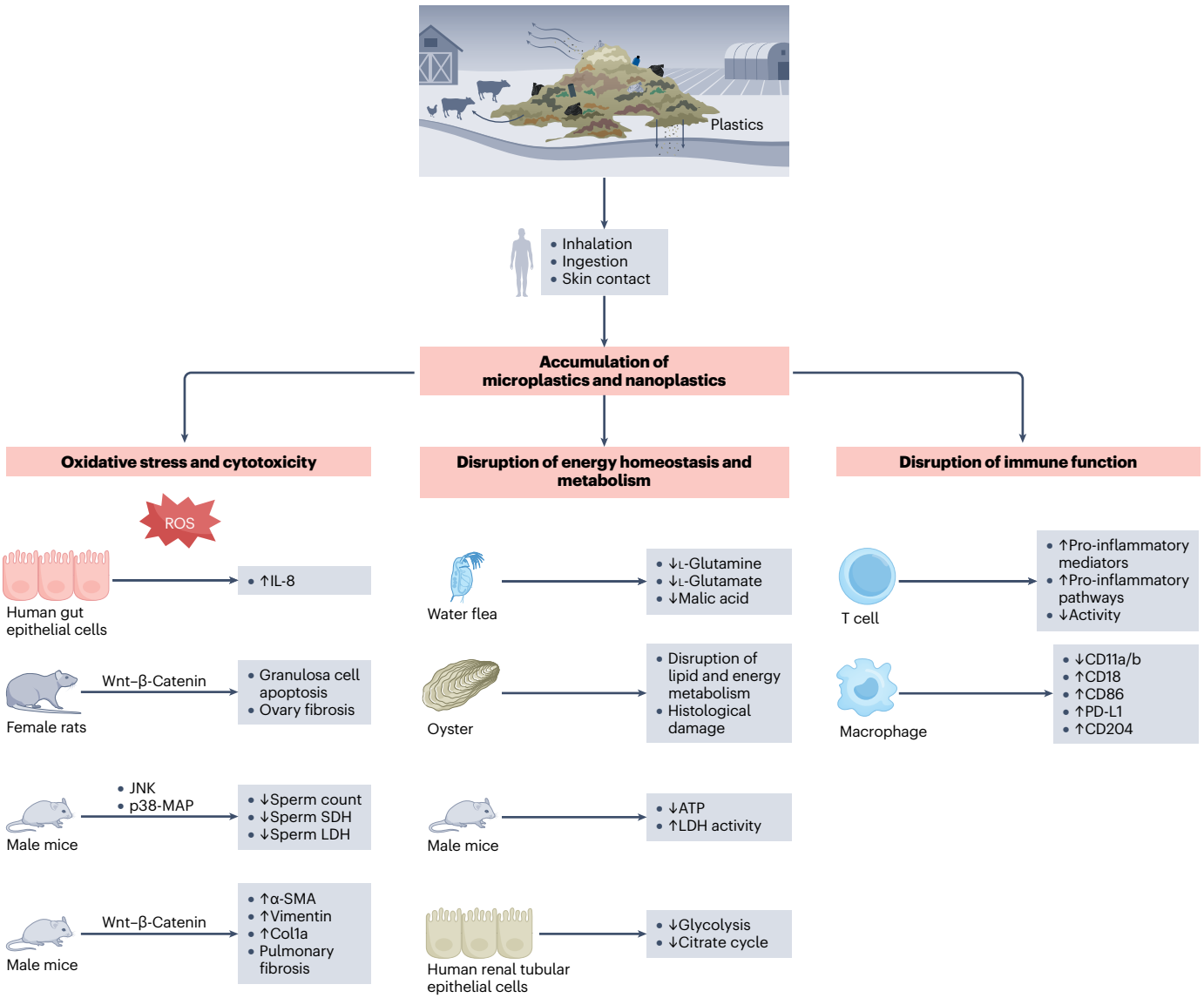

Fig. 1 | The toxic effects of microplastics and nanoplastics. Microplastics (MPs) and nanoplastics (NPs) are ubiquitous environmental pollutants that contaminate water, air and the food chain. They can enter the bodies of humans or animals via ingestion, inhalation or skin contact[19]. Studies in human cells and animal models have shown that these particulate plastics accumulate in tissues[27] and have toxic effects owing to induction of oxidative stress[36,38,40,41] and cytotoxicity[39], disruption of energy metabolism[12,47,48] and disruption of immune function[56]. In human gut epithelial cells, exposure to MPs had cytotoxic effects, including increased production of IL-8 (ref. 39). In female rats, oral exposure to MPs induced oxidative stress, granulosa cell apoptosis and ovary fibrosis[41], whereas in male mice, oral exposure to MPs resulted in oxidative stress, reduced sperm count and reductions in the activity of succinate dehydrogenase (SDH) and lactate dehydrogenase (LDH) in sperm[40]. Inhalation of MPs, led to oxidative stress, pulmonary fibrosis and increases in the expression of α-smooth muscle actin (αSMA), vimentin and collagen 1a (Col1a) in the lungs of male mice[38]. Reported metabolic effects of MPs and NPs include downregulation of L-glutamine, L-glutamate and malic acid in water fleas[47], disruption of lipid and energy metabolism in oysters[48], and a reduction in ATP and an increase in LDH activity in male mice[12]. In renal tubular epithelial cells, exposure to NPs led to metabolic changes, including downregulation of glycolysis and the citrate cycle[50]. Exposure to MPs and NPs has led to upregulation of pro-inflammatory mediators and pathways in various models. MPs and NPs have also been shown to suppress T cell activity[56] and to alter the expression of cell surface markers with roles in the immune response of macrophages[59]. ROS, reactive oxygen species.

Particulate plastics that enter the circulatory system can translocate to and accumulate in various organs[25]. In mice that were exposed to fluorescent MPs (5 μm and 20 μm) by oral gavage, MPs accumulated in the gut, liver and kidney and the pattern and kinetics of this accumulation were dependent on particle size[12]. Another study, in which polystyrene (PS)-MPs (5 μm) and mixed polymer MPs consisting of PS, polyethylene (PE) and poly-(lactic-co-glycolic acid) (1–5 μm) were orally administered to mice, reported that these particles crossed the intestinal barrier, entered the bloodstream, and accumulated in the kidney, liver and brain[26].

In humans, varying concentrations of MPs have been identified in a wide range of tissues and body fluids, including lung, liver, kidney, spleen, placenta, hair, skin, blood, saliva, sputum, breast milk, bronchoalveolar lavage fluid, brain, meconium and faeces[14,27]. The digestive and respiratory tracts — the primary entry points for ingested and inhaled MPs — typically exhibit the highest MP concentrations. The predominant route of MP exposure is oral ingestion, with an estimated annual intake ranging from 39,000 to 52,000 particles per person[28]. Inhalation is also an important exposure pathway, with an estimated daily uptake via this route of approximately 272 particles per person[27,28]. The liver and spleen accumulate MPs via blood filtration. An analysis of blood samples from healthy volunteers reported a mean MP concentration of 1.6 μg/ml[29]. Other studies have reported MP abundances of 0–13 particles per sample or 3.2 particles per gram in cirrhotic liver tissue[30] and 31 MPs per sample in lung tissue obtained at autopsy from adults who were non-smokers[31]. In addition, small MPs can cross the blood–brain barrier under certain conditions[24]. A study that used micro-Raman spectroscopy to detect MPs in healthy kidney tissue from 10 patients undergoing nephrectomy and urine samples from 10 healthy donors identified 66 particles, of which 26 were characterized. These particles ranged in size from 1 to 29 μm in the kidneys and 3 to 13 μm in the urine. The most common polymers were PE and PS[32].

The presence of particulate plastics in faeces has become a key focus in environmental research given the ability to assess exposure levels in various organisms, including humans. Faeces have been shown to transport particulate plastics between ecosystems and disperse them into aquatic and terrestrial environments, extending their lifespan and potential bioavailability[33]. A study that included 24 male participants from Beijing, China, reported that 95.8% had MPs in their faeces. The concentration of MPs in the faecal samples ranged from 1 to 36 particles per gram and the total mass per person ranged from 0.01 to 14.6 mg. The most common type of MP, polypropylene, was found in 95.8% of the samples. A moderate correlation ($r = 0.445$, $P = 0.029$) was found between the amount of packaged water or beverage consumed and the abundance of faecal MPs, demonstrating that diet influences the presence of MPs in humans[34].

Pathways of particulate plastic toxicity

The toxicity pathways of particulate plastics involve various mechanisms, including physical accumulation in tissues leading to blockages or disruptions and chemical toxicity from leached additives or absorbed pollutants. Smaller particles can infiltrate biological systems more deeply, resulting in oxidative stress[35]. Particulate plastic pollution can also have cytotoxic effects and disrupt energy homeostasis, metabolism and immune function (Fig. 1).

Oxidative stress and cytotoxicity

The cellular toxicity of MPs and NPs involves their ability to penetrate cells. MPs can inflict cellular damage through mechanical impact or by prompting cells to generate reactive oxygen species[36]. Four principal cytotoxic mechanisms of MPs have been described: induction of oxidative stress, disruption of organelle membranes, initiation of immune responses and disruption of energy homeostasis and metabolism.

In juvenile rats, 28 days of oral exposure to 1,000 nm PS-MPs (2.0 mg/kg per day) resulted in intestinal damage and reductions in the rate of increase in body weight and organ indexes of the kidney, heart and ovary[37]. In the kidney, PS-MP exposure induced endoplasmic reticulum (ER) stress and inflammatory responses, which were likely mediated by oxidative stress. Administration of the anti-oxidant N-acetylcysteine, alleviated PS-MP-induced oxidative stress, ER stress, renal inflammation and apoptosis, suggesting that the nephrotoxicity of PS-MPs in juvenile rats is primarily due to induction of oxidative damage and ER stress.

Another study investigated the impact of inhalation of 5 μm PS-MPs on pulmonary fibrosis in mice[38]. The PS-MPs induced pulmonary fibrosis in a dose-dependent manner, with higher doses resulting in significant increases in the expression of α-smooth muscle actin (αSMA), vimentin and collagen 1a (Col1a). Immunofluorescence assays showed reduced levels of surfactant protein-C (SP-C) and increased Krebs von den lungen-6 (KL-6) in the lung tissues of mice that had inhaled PS-MPs compared with controls, indicating alveolar epithelial injury. Inhalation of PS-MPs also resulted in reductions in the levels of superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) in the lungs, indicating oxidative stress. These findings suggest that inhalation of PS-MPs can lead to pulmonary fibrosis owing to oxidative stress and activation of the Wnt–β-catenin pathway.

PS-MPs (4.8 to 5.8 μm) were also shown to have cytotoxic effects in a human gut epithelial cell line (HRT-18) and a mouse intestinal cell line (CMT-93) and to increase oxidative stress in CMT-93 cells[39]. Magnetic bead multiplex assays and gene expression analyses showed that exposure of HRT-18 cells to PS-MPs increased their release of IL-8, suggesting that these particles induce a pro-inflammatory response that could contribute to their cytotoxicity.

The reproductive toxicity of PS-MPs (5.0–5.9 μm) was examined in male BALB/c mice[40]. Six weeks of exposure to PS-MPs resulted in significant decreases in sperm count and motility, an increase in sperm deformity and a reduction in the activity of sperm metabolism-related enzymes (succinate dehydrogenase and lactate dehydrogenase) and serum testosterone levels. These effects were associated with oxidative stress and activation of the c-Jun N-terminal kinase and p38-mitogen-activated protein kinase (MAPK) pathways. In female rats, PS-MPs induced granulosa cell apoptosis owing to oxidative stress and ovary fibrosis owing to activation of the Wnt–β-catenin signalling pathway[41].

MPs and NPs have also been shown to induce oxidative stress in endothelial cells and promote inflammatory responses in multiple cell types[42,43]. In response to foreign particles, immune cells activate NADPH oxidase (NOX) and other enzymes to produce reactive oxygen species, triggering oxidative cascades[44]. Notably, excessive reactive oxygen species generation due to NOX activation has been implicated in the development of cardiovascular disease (CVD)[45,46]. This finding suggests that activation of NOX by particulate plastics is a potential mechanism by which these particles might increase the risk of CVD.

Disruption of energy homeostasis and metabolism

Experimental evidence suggests that particulate plastics can disrupt energy homeostasis and metabolism. In the water flea *Daphnia magna*, exposure to PE-MPs of 20 μm (PE-MPs-20) and 30 μm (PE-MPs-30) in size

resulted in gut accumulation and decreases in heart rate and survival. A metabolomic analysis identified significant changes in 75 metabolites, with PE-MPs-20 affecting phospholipid metabolism by regulating phosphatidylcholine and phosphatidylethanolamine and PE-MPs-30 disrupting energy metabolism by downregulating metabolites such as L-glutamine, L-glutamate and malic acid[47].

A study in oysters (*Crassostrea gigas*) showed that 21 days of exposure to 10 μg/l and 1,000 μg/l concentrations of PE-MPs (36.72 μm) and polyethylene terephthalate (PET)-MPs (31.11 μm) resulted in accumulation of MPs in the gills and digestive glands, disruption of lipid and energy metabolism and histological damage. The toxic effects were more pronounced at higher MP concentrations and PET-MPs had greater toxic effects than PE-MPs. A metabolomic analysis showed that MP exposure induced significant changes in metabolic profiles, indicating alterations in energy metabolism and inflammatory responses and highlighting the ecological risks of MPs in marine environments[48].

Male mice that were exposed to 5 μm and 20 μm PS-MP at concentrations of 0.01, 0.1 and 0.5 mg per day for 4 weeks exhibited significant reductions in liver total cholesterol and triglyceride levels, as well as dose-dependent changes in biological parameters related to energy and lipid metabolism, including a decrease in ATP and an increase in lactate dehydrogenase activity[12]. We found in mice that PS-MP exposure disrupted hepatic lipid metabolism and energy homeostasis by activating the nuclear receptor subfamily 4 group A member 1 (NR4A1)–AMP-activated protein kinase (AMPK) signalling cascade[49]. Exposure to PS-MPs also resulted in decreases in serum triglyceride and increases in reactive oxygen species in liver tissue.

An integrated proteomic and metabolomic approach was used to investigate the impact of PS-NPs (80 nm) on a human renal tubular epithelial cell line (HKC). The results showed significant changes in 785 proteins and 17 metabolites with roles in metabolic pathways such as glycolysis and the citrate cycle. A seahorse analysis confirmed that the PS-NPs induced changes in energy metabolism. The PS-NPs also inhibited mTOR complex 1 (mTORC1) signalling, providing further evidence that NP exposure causes metabolic dysfunction[50].

Disruption of immune function

MPs and NPs can interact with immune cells, alter the balance of the immune system and cause tissue damage through the production of reactive oxygen species[51,52], activation of Toll-like receptors (TLRs) and induction of inflammatory cytokines[53]. Various MPs and NPs have also been shown to activate the NLRP3 inflammasome, NFκB signalling and other pro-inflammatory pathways[54,55]. MPs and NPs have also been shown to suppress T cell activity and alter immune regulation[56,57]. Single-cell RNA sequencing of gut and brain tissues from mice that were fed fabricated NPs (~500 nm) and MPs (~2 μm) demonstrated that NPs activate intestinal macrophages more efficiently than MPs[58]. The NPs induced lysosomal damage in the intestinal macrophages, leading to production of IL-1 and the induction of systemic immune responses. In addition, Intestinal IL-1 signalling led to activation of microglia and enhanced differentiation of T helper type 17 (Th17) cells in the brain, which likely contributed to cognitive impairment and memory loss in the NP-fed mice. These findings suggest a gut–brain axis-mediated mechanism by which NPs impair brain function.

Another study showed that PS-MPs (ranging in size from 100 nm to 6 μm) were internalized by and altered the functions of cultured macrophages in a size- and dose-dependent manner, inducing oxidative stress and altering the expression of cell surface markers with roles in the immune response such as CD11a/b, CD18, CD86, PD-L1 and CD204 (ref. 59). Larger particles induced more substantial changes than smaller particles, with greater effects in macrophages that had absorbed higher numbers of particles. These findings suggest that high levels of exposure to PS-MPs may lead to the generation of macrophage subpopulations with impaired functions, potentially compromising the innate immune response.

In mice, repeated oral administration of PE-MPs for 90 days led to an increase in the blood neutrophil count and significant reductions in the total white blood cell and lymphocyte levels compared with the control mice[60]. The researchers also found that PE-MP particles migrated to the surface of mast cells in the gastric tissue and splenic cells exhibited organelle abnormalities, including accumulation of mitochondria, suggesting potential immune system and cellular dysfunction. Although human studies are limited, the available data suggest that MPs and NPs can also disrupt the gut microbiota and intestinal function, which may promote the development of chronic immune disorders[61].

Interactions with other contaminants

Particulate plastics seldom occur in isolation but are often accompanied by other hazardous substances, such as toxic metal ions, persistent organic pollutants and fungicides in natural ecosystems, potentially leading to synergistic or combined toxicity[62–64]. The adsorption of toxic chemicals onto particulate plastics depends on the particle size, surface characteristics and porosity. This adsorption could lead to the accumulation of toxins in the food chain, resulting in a persistent hazard to human health[65].

In addition to being absorbed from the surrounding environment, toxic metals are often incorporated into plastic products as additives or fillers. The concentrations of heavy metals detected in particulate plastics range from <1 to <200,000 μg/g[66]. Furthermore, in a zebrafish embryo model, the toxic effects of PS-NPs (50 nm) were exacerbated by the presence of Au ions. Compared with embryos that were exposed to PS-NP alone, those that were exposed to both PS-NPs and Au ions showed increased reactive oxygen species generation, pro-inflammatory responses and mitochondrial damage[67].

Particulate plastics and the kidney

Evidence from in vitro experiments and animal studies suggests that particulate plastics have toxic effects on the kidney (Table 1). Importantly, patients on long-term kidney replacement therapy are at risk of increased exposure to particulate plastics, potentially owing to contamination of medical devices and dialysis fluids. Further research is required to investigate the mechanisms of particulate plastic absorption, systemic distribution, renal accumulation and excretion, as well as the potential impacts of these pollutants on kidney health.

Impact of particulate plastics on kidney health

Particulate plastics can induce cellular and molecular alterations in the kidney. Exposure of human embryonic kidney (HEK293) cells to 1 μm PS-MPs reduced their proliferation and altered their morphology but did not substantially affect their viability. The HEK293 cells internalized the PS-MPs, increased reactive oxygen species production and downregulated expression of the glycolytic enzyme glyceraldehyde-3-phosphate dehydrogenase and the anti-oxidant enzymes SOD2 and catalase[68]. Similarly, we found that exposure of human kidney proximal tubular epithelial cells (HK-2 cells) to 2 μm PS-MPs resulted in uptake of the PS-MPs and increases in ER stress, inflammation and levels of mitochondrial reactive oxygen species[69]. PS-MPs also induced autophagy of the HK-2 cells by altering the activity of the AKT–mTOR

Table 1 | The effects of particulate plastics on the kidney

Study	Model	Intervention	Findings	Ref.
Goodman et al. (2022)	HEK293 cells	PS-MPs (1 µm)	Exposure to PS-MPs resulted in a substantial increase in intracellular reactive oxygen species levels and suppressed the expression of key metabolic and anti-oxidant enzymes, including GAPDH, SOD2 and catalase	68
Wang et al. (2021)	HK-2 cells	PS-MPs (2 µm)	Exposure to PS-MPs resulted in increases in ER stress, autophagy-related proteins, inflammation and levels of mitochondrial reactive oxygen species	69
	Male C57BL/6 mice		After oral treatment with PS-MPs, kidneys showed histopathological lesions and increased levels of ER stress, inflammatory markers and autophagy-related proteins	
Wang et al. (2024)	HK-2 cells	PS-MPs (2 µm)	PS-MP exposure markedly enhanced EV biogenesis and upregulated the expression of ER stress-associated proteins	70
	Mouse fibroblasts		PS-MP exposure led to reactive oxygen species production, induced ER stress markers and increased the expression of profibrotic proteins	
	Male C57BL/6 mice		PS-MP exposure led to reactive oxygen species production in kidney tissues and increased the expression of EV-related markers in urine	
Chen et al. (2022)	HEK293 cells	PS-MPs (3.54±0.39 µm)	PS-MPs had strong affinity for and were internalized by HEK293 cells, triggering oxidative stress and cytotoxicity via downregulation of HO-1	71
Meng et al. (2022)	Chickens	PS-MPs (5 µm)	Oral treatment with PS-MPs caused mitochondrial dysfunction, increased levels of pro-inflammatory factors, induced necroptosis and increased levels of oxidative stress in kidneys	72
Xiong et al. (2023)	Male C57Bl/6J mice	PS-MPs (80 nm, 0.5 µm and 5 µm)	Oral treatment with PS-MPs induced oxidative stress, inflammation and apoptosis in the kidney, resulting in severe kidney damage and fibrosis	73
Meng et al. (2022)	Mice	PS-NPs (50 nm) and PS-MPs (300 nm, 600 nm and 4 µm)	600 nm PS-MPs had greater biological toxicity than other particle sizes, resulting in greater mortality, reductions in kidney weight, oxidative stress, inflammation and kidney injury	76
Liang et al. (2024)	Male C57BL/6 mice	PS-MPs (2 µm)	Treatment with PS-MPs impaired gut barrier integrity, increased renal C5aR expression and urinary C5a levels and led to reduced kidney function, proteinuria and progressive renal fibrosis	77

C5a, complement component 5a; C5aR, C5a receptor; ER, endoplasmic reticulum; EV, extracellular vesicle; GADPH, glyceraldehyde-3-dehydrogenase; HEK293, human embryonic kidney 293; HK-2, human kidney proximal tubular epithelial cells; HO-1, haem oxygenase 1; SOD2, superoxide dismutase 2.

and MAPK signalling pathways. Furthermore, in mice, oral administration of PS-MPs led to increases in kidney histopathological lesions, ER stress and inflammation.

We also showed that exposure to PS-MPs (2 µm) increased extracellular vesicle (EV) production and induced ER stress-related proteins in human renal tubular cells (HK-2 cells) without inducing inflammation-related proteins[70]. Exposure to PS-MP or to EV-containing conditioned medium from HK-2 cells that had been treated with PSMPs also increased the production of ROS, ER stress proteins and fibrosis-related proteins in fibroblasts. Further analysis suggested a role of the autophagy-related protein beclin 1 in increasing the expression of EV-related markers such as CD63 and CD81 in renal tubular cells following PS-MP treatment. In a mouse model, oral administration of PS-MPs led to increased urinary expression of an EV marker (CD63). These findings suggest that induction of EVs by PS-MPs contributes to kidney damage via mechanisms involving ER stress, oxidative stress and fibrosis. Furthermore, EVs produced by kidney cells in response to particulate plastics could potentially impact other cell types throughout the body.

Another study showed that PS-MPs (3.54 ± 0.39 µm) can adhere to and be absorbed by HEK293 cells, resulting in oxidative stress and cytotoxicity by inhibiting the anti-oxidant haem oxygenase-1 (ref. 71). Non-cytotoxic levels of PS-MPs (3 ng/ml) induced inflammatory cytokines, whereas cytotoxic levels of PS-MPs (300 ng/ml), induced autophagy, which potentially attenuated inflammation by downregulating NLRP3 expression. Cytotoxic levels of PS-MPs also reduced the levels of α1-antitrypsin and zonula occludens-2 proteins in the HEK293 cells, suggesting that exposure to these particles may weaken the integrity of the kidney barrier and increase the risk of acute kidney injury.

In chickens, oral intake of 5 µm PS-MPs caused renal damage, which increased in severity with dose (1, 10, or 100 mg/l)[72]. In the kidney, the PS-MPs caused mitochondrial dysfunction, with structural damage and dysregulation of proteins with roles in mitochondrial dynamics (MFN1/2, OPA1 and Drp1), and increased levels of oxidative stress, as evidenced by decreases in anti-oxidant enzymes. Histopathological analysis showed kidney damage and inflammation linked to NFκB P65 activation and increased levels of pro-inflammatory factors (TNF, inducible nitric oxide synthase, IL-1β and IL-6). In addition, the PS-MPs induced necroptosis in kidney tissues via the receptor interacting protein kinase-1 (RIP1)–receptor interacting protein kinase (RIP3)–mixed lineage kinase domain-like protein (MLKL) pathway.

Mice that were fed various sizes of PS-MPs (80 nm, 0.5 µm and 5 µm) exhibited kidney damage[73]. PS-MPs induced oxidative stress, inflammation and apoptosis in the kidney, resulting in severe kidney damage and fibrosis. Transcriptome analysis showed that long-term exposure to 80 nm PS-MPs resulted in alterations in the expression of genes involved in immune responses, whereas long-term exposure to 0.5 µm and 5 µm PS-MPs resulted in alterations in genes with roles in circadian rhythm. These results highlight the complex mechanisms by which particulate plastics contribute to renal injury.

The presence of MPs is notably prominent in fast food, takeout, beverages, and high-fat diets (HFDs) owing to contamination at various

stages, including production, processing, packaging and storage. In mice, treatment with PS-MPs (1 µm) plus an HFD exacerbated glomerular damage, tubular damage and tubulointerstitial fibrosis compared with treatment with either an HFD or PS-MPs alone[74]. Single-cell transcriptomic profiling of kidney tissues showed increased activation of the IL-17 signalling pathway, MAPK signalling cascade and oestrogen receptor signalling network in mice subjected to PS-MPs plus an HFD compared with those that received an HFD alone. In renal epithelial cells, the PS-MPs and HFD activated pathways that are associated with inhibition of renal development and reactive oxygen species-driven tumorigenesis. Exposure to PS-NPs has also been shown to increase lipopolysaccharide (LPS)-induced apoptosis in mouse kidneys and HEK293 cells via induction of ER stress, activation of the oxidative stress-regulated inositol-requiring enzyme 1 (IRE1)–X-box binding protein 1 (XBP1) pathway and upregulation of caspase 3 and caspase 12 (ref. 75).

Another study showed that when PS-NPs (50 nm) and PS-MPs of various sizes (300 nm, 600 nm and 4 µm) were administered to mice, the 600-nm particles aggregated and the zeta potential of all particles increased during digestion in the gastrointestinal tract, indicating changes in surface charge and stability[76]. All sizes of particles accumulated in the kidneys, but the 600 nm PS-MPs had the greatest biotoxicity, resulting in kidney weight loss, increased mortality, kidney damage, oxidative stress, and inflammation.

In mice, exposure to 2 µm PS-MPs compromised gut barrier integrity and led to elevated renal C5a receptor (C5aR) expression and increased urinary levels of the complement component C5a, resulting in reduced kidney function, increased proteinuria and progressive renal fibrosis[77]. Restoring the gut barrier using antibiotics not only alleviated kidney injury but also reduced urinary C5a levels and renal C5aR expression, indicating a role of the gut–kidney axis in PS-MP-induced kidney injury.

Several studies have provided evidence that natural products might mitigate particulate plastic-induced damage in animal models, but research on their role in preventing kidney and cardiovascular damage is limited (Supplementary Table 1). One study reported that treatment with the natural flavonoid ginkgetin reduced oxidative stress, inflammation and kidney damage in rats that were exposed to PS-MPs (0.01 mg/kg)[78]. This area of research warrants further investigation.

Combined kidney toxicity of particulate plastics and other environmental contaminants

Environmental contaminants such as heavy metals and polycyclic aromatic hydrocarbons (PAHs) enhance the toxic effects of MPs and NPs on kidney cells[79–81]. For example, the cytotoxic effects of exposure to PS-NPs of various sizes (20, 60, 100, 500 and 1,000 nm) on human embryonic kidney 293 T cells were found to be substantially increased when the PS-NPs were enriched with lead (Pb^{2+})[79]. Compared with exposure to PS-NPs alone, exposure to PS-NPs plus Pb^{2+} resulted in increases in reactive oxygen species production, inflammatory responses, membrane damage, mitochondrial dysfunction and a reduction in ATP levels.

A study in which mice were exposed to 5 µm PS-MPs and 50 mg/l cadmium (Cd) in water for 3 months showed that the presence of PS-MPs exacerbated Cd-induced kidney damage by increasing oxidative stress, autophagy, apoptosis and fibrosis[80]. Similarly, in mouse kidneys and HEK293 cells, exposure to di(2-ethylhexyl) phthalate (DEHP) and PS-MPs (1-10 µm) significantly increased oxidative stress and autophagy compared with exposure to either pollutant alone, as evidenced by increases in the expression of genes related to the reactive oxygen species–AMPK–serine/threonine-protein kinase ULK1 (ULK1) and peroxisome proliferator-activated receptor-γ coactivator 1α (Ppargc1α)–mitofusin 2 (Mfn2) signalling pathways and upregulation of mRNA and protein levels of autophagy markers[81]. These findings highlight the potential kidney health risks associated with the coexistence of particulate plastics and other environmental pollutants.

Dialysis-related particulate plastic exposure

Individuals with kidney failure might be exposed to particulate plastics during dialysis procedures, which involve extensive contact between their blood, the dialysate and the dialysis equipment. Furthermore, as patients with kidney failure have limited or no urine output, they may be at higher risk of accumulating particulate plastics than individuals with normal kidney function.

Although the presence of particulate plastics in dialysis fluids has not been investigated, the prevalence of particulate plastics in water systems suggests that the water used in these fluids, which is derived from surface water or groundwater supplied directly or via distributors, could be contaminated[82,83]. Further studies are needed to investigate this risk. Water treatment technologies, including membrane filtration in drinking water treatment plants, could also be sources of particulate plastics if the membranes are damaged[34].

The use of polyvinyl chloride (PVC) containing phthalate plasticizers such as DEHP in dialysis apparatus is also a concern. These plasticizers, which are added to increase flexibility, lack covalent bonds with PVC and pose substantial health risks because they can leach into the patient's blood during the dialysis session[84]. In mice, combined exposure to MPs and DEHP was shown to delay skin healing[85]. Compared with exposure to either contaminant alone, combined exposure to MPs and DEHP increased oxidative stress and promoted the release of neutrophil extracellular traps, which led to activation of the NFκB pathway and inhibition of fibrosis. Another mouse study showed that combined exposure to DEHP and PS-MPs induced oxidative stress and renal autophagy via activation of the reactive oxygen species–AMPK–ULK1 pathway[81]. The toxicity of DEHP and PS-MPs exhibited a synergistic effect, with combined exposure resulting in greater kidney damage than exposure to either PS-MPs or DEHP alone. This effect was likely mediated by upregulation of cytochrome P450 1A1 (CYP1A1) and cytochrome P450 1B1 (CYP1B1), leading to excessive reactive oxygen species-induced autophagy.

Potential strategies to mitigate particulate plastic contamination in dialysis settings include regularly maintaining and monitoring dialysis equipment and improving water treatment processes (Supplementary Table 2). Use of advanced water treatment systems and high-efficiency particulate arresting filters might also reduce the risk of particulate plastic contamination. Regular MP-failure mode and effect analysis (MP-FMEA) assessments in wastewater treatment plants could potentially mitigate contamination of water sources[86]. According to the WHO, reverse osmosis membranes are adequate for removing MPs and NPs; nanofiltration techniques can remove particles measuring >0.001 µm, whereas ultrafiltration techniques effectively eliminate particles measuring >0.01 µm[87]. Protecting patients on dialysis from particulate plastic exposure requires collaborative efforts from health care providers, researchers and policymakers.

Particulate plastics and the cardiovascular system

MPs and NPs have been identified as potential risk factors for CVD[17,88,89]. In the past decade, experimental studies have reported that the cardiac effects of particulate plastics include heart rate alterations, pericardial oedema, cardiac function impairment and myocardial fibrosis, whereas

their toxic effects at (micro)vascular sites include haemolysis, thrombosis, blood coagulation and damage to vascular endothelial cells[88]. Although these findings have contributed to our understanding of the potential cardiovascular toxicity of particulate plastics, substantial knowledge gaps persist regarding the underlying mechanisms and potential human health risks.

Impact of particulate plastics on the cardiovascular system

Exposure of human vascular endothelial cells (EA.hy926 cells) to PS-MPs at a concentration that could occur in the blood (4×10^{-6}–40 μg/ml) resulted in oxidative stress, apoptosis and depletion of zonula occludens-1 protein (ZO-1), resulting in vascular barrier dysfunction[90]. However, exposure to PS-MPs did not induce a substantial increase in the expression of lectin-like oxidized low-density lipoprotein receptor-1 (LOX-1), suggesting that these particles do not have a role in the pathogenesis of atherosclerosis.

Another study showed that patients with vascular calcification, which is a risk factor for poor cardiovascular disease prognosis, had higher levels of particulate plastics in their faeces than patients without vascular calcification[91]. Furthermore, the thoracic aortic calcification score significantly correlated with the abundance of particulate plastics in faeces. As excretion in faeces is an essential clearance mechanism for particulate plastics, their abundance in faeces could potentially be used as a measure of particulate plastic exposure in cardiovascular disease risk assessment[22,91].

MPs or NPs can interact with blood cells, such as platelets, forming aggregated complexes that may induce vascular obstruction and compromise cardiac function[92]. Exposure of whole-blood samples to PS-NPs (0.05 and 0.1 μm) resulted in toxic effects on white blood cells, but uptake and effects varied between cell types. Following this exposure, monocytes and polymorphonuclear cells, but not lymphocytes, exhibited increased DNA damage and changes in cytokine expression related to inflammation and immune responses[93]. Another study that investigated the effects of PS-NPs (100 nm) on human blood cells showed that coronated NPs induced protein conformational changes and denaturation, which enhanced their genotoxic and cytotoxic effects compared with virgin NPs[94].

Exposure of human umbilical vein endothelial cells (HUVECs) to PS-MPs (0.5 μm) resulted in dose-dependent intracellular accumulation of the particles and significantly reduced cell viability and tube formation, which are crucial for angiogenesis[95]. PS-MPs also inhibited angiogenic signalling, wound healing and cell migration. Short-term exposure (6 h) to PS-MPs impaired the ability of the HUVECs to form vascular structures, whereas prolonged exposure (48 h) induced autophagy and necrosis.

A human pluripotent stem cell-derived 3D cardiac organoid model was used to investigate the effects of PS-MPs on the heart[96]. Following exposure to PS-MPs (0.025 μg/ml, 0.25 μg/ml and 2.5 μg/ml) PS-MPs, the organoids showed increased levels of oxidative stress, inflammatory responses, apoptosis and collagen deposition. They also showed significant alterations in the expression of genes related to hypertrophy (*MYH7B*, *ANP*, *BNP* and *COL1A1*) and increased expression of cardiac-specific markers (*MYL2*, *MYL4* and *CX43*), suggesting that PS-MPs may trigger cardiac hypertrophy.

NPs can more easily cross the blood–brain and blood–placenta barriers and have greater toxic effects on target organs than MPs[51]. Exposure to PS-NPs (20 nm) was shown to reduce the efficiency of cardiomyocyte differentiation from human embryonic stem cells (hESCs), leading to the generation of immature cardiomyocytes and smaller cardiac organoids exhibiting impaired contractility[97]. PS-MPs reduced the self-renewal and pluripotency of hESCs, increased mitochondrial oxidative stress, activated the p38–ERK MAPK signalling pathway and inhibited autophagic flux. The dose of PS-MPs applied in the study (5 μg/ml) was comparable with the concentration of particulate plastics reported in human blood (1.6 μg/ml), demonstrating the potential of use of hESCs to assess the impact of particulate plastic exposure on cardiac development[29,97]. These studies offer novel insights into the risk assessment of human cardiovascular and embryogenic defects arising from environmental MP and NP pollution.

A prospective, multicentre, observational study in 304 patients who underwent carotid endarterectomy for asymptomatic carotid artery disease provided clinical evidence of a role of particulate plastics as a potential risk factor for cardiovascular disease[17] (Table 2). PE and PVC, important components of particulate plastics, were detected in carotid artery plaque in 58.4% and 12.1% of patients, respectively, with mean levels of PE of 21.7 ± 24.5 μg/mg of plaque and PVC of 5.2 ± 2.4 μg/mg of plaque. During 34 months of follow-up, patients with carotid artery plaques that contained PE or PVC had an increased risk of myocardial infarction, stroke or death from any cause compared with those in whom particulate plastics were not detected.

Role of autophagy dysfunction in particulate plastic-induced cardiovascular toxicity

The available data suggest that the principal mechanisms that underlie particulate plastic-induced cardiovascular toxicity involve oxidative stress, inflammation, apoptosis, pyroptosis and interactions between particulate plastics and various cellular components such as mitochondria[88]. However, emerging evidence suggests that autophagy dysfunction might also have a role.

Autophagy is an evolutionarily conserved lysosomal pathway that is essential for degrading damaged organelles and detrimental protein aggregates, thereby preserving cellular homeostasis and viability. Conversely, dysregulation of autophagy can precipitate cellular injury and death, thereby fostering pathological cascades, including endothelial dysfunction, vascular ageing and cardiovascular pathologies, including atherosclerosis and vascular calcification[98,99]. Autophagy also has roles in angiogenesis and in the paracrine regulation of vasoactive substances from the endothelium[98]. The critical signalling pathways involved in the reciprocal modulation between autophagy and CVD include the PI3K–Akt–mTOR, IGF–EGF, AMPK–mTOR, Wnt–β-catenin, MAPK, Nrf2–p62, p53 and NFκB pathways[99].

A role of autophagy in various types of nanoparticle-induced toxicity has been shown in various experimental systems[100–102]. However, to date, few studies have provided evidence of an involvement of autophagy in MP or NP-induced toxicity in the cardiovascular system. One study demonstrated that 100 nm PS-NPs, but not 500 nm PS-NPs, could be internalized by HUVECs, accumulate in the cytoplasm and induce autophagosome formation along with blockade of autophagic flux[103]. Notably, the reactive oxygen species pathway and inflammatory response did not serve as upstream mechanisms for the autophagy dysfunction induced by 100 nm PS-NPs in HUVECs, necessitating further elucidation of the underlying mechanisms. As mentioned above, PS-NPs (20 nm) have also been shown to block autophagic flux in hESCs[97]. In this study, PS-NPs exposure initiated the formation of autophagosomes, as evidenced by elevated levels of the autophagy-related protein LC3-II/I and autophagy protein 5 (Atg5), but also induced increased p62 expression and decreased expression of lysosome-associated membrane glycoprotein 1 (Lamp1), suggesting

Table 2 | The effects of particulate plastics on the cardiovascular system

Study	Sample or model	Intervention	Findings	Ref.
Marfella et al. (2024)	Carotid artery plaque from patients with asymptomatic carotid artery disease	NA	PE and PVC were identified in carotid artery plaques in 58.4% and 12.1% of patients, respectively; the presence of these plastics was associated with an increased risk of myocardial infarction, stroke and all-cause mortality	17
Chen et al. (2023)	EA.hy926 cells	PS-MPs (3.9±0.9 μm)	Exposure to PS-MPs led to oxidative stress, apoptosis and ZO-1 depletion, resulting in vascular barrier dysfunction	90
Yan et al. (2023)	Faeces	NA	Patients with vascular calcification had higher levels of particulate plastics in their faeces than those without vascular calcification	91
Ballesteros et al. (2020)	White blood cells	PS-NPs (0.05 and 0.1 μm)	Exposure to PS-NPs resulted in DNA damage and changes in pro-inflammatory cytokines in monocytes and polymorphonuclear cells but not in lymphocytes	93
Lee et al. (2021)	HUVECs	PS-MPs (0.5 μm)	Exposure to PS-MPs reduced cell viability, angiogenic tube formation, angiogenic signalling, wound healing and cell migration	95
Zhou et al. (2023)	Human pluripotent stem cell-derived cardiac organoids	PS-MPs (1 μm)	Exposure to PS-MPs resulted in increased levels of oxidative stress, inflammatory responses, apoptosis and collagen deposition	96
Li et al. (2024)	hESCs	PS-NPs (20 nm)	Exposure to PS-NPs reduced hESC self-renewal and pluripotency, induced mitochondrial oxidative stress, activated the p38-ERK–MAPK pathway, inhibited autophagy and impaired cardiomyocyte differentiation, resulting in immature cardiomyocytes and smaller, less contractile cardiac organoids	97

EA.hy926, human vascular endothelial cell line; hESCs, human embryonic stem cells; HUVECs, human umbilical vein endothelial cells; NA, not applicable; ZO-1, zonula occludens-1 protein.

that a defect in the fusion between autophagosomes and lysosomes led to the suppression of autophagic flux. This finding demonstrates that autophagy dysfunction contributes to PS-NP-induced defects in the pluripotency of hESCs.

Combined cardiovascular toxicity of particulate plastics and other environmental contaminants

Most of the existing data concerning the combined cardiovascular toxicity of particulate plastics and other environmental contaminants have been obtained in studies utilizing fish models. These effects can be categorized into synergistic, antagonistic, biphasic and other effects[45]. For example, exposure of zebrafish embryos to PS-NPs in conjunction with azole fungicides, chloroauric acid (HAuCl4) or 2,2′,4,4′-tetrabromodiphenyl ether (BDE-47), exacerbated cardiovascular damage compared with exposure to PS-NPs alone, resulting in severely underdeveloped hearts, pericardial oedema and reduced heart rate[63,64,104]. The underlying mechanisms of synergistic cardiovascular toxicity potentially include increases in reactive oxygen species, lipid peroxidation, apoptosis, pro-inflammatory responses and mitochondrial damage[63,64,104].

The presence of particulate plastics has also been shown to have antagonistic effects in mitigating the cardiovascular toxicity of PAHs and metal ions. Exposure of zebrafish larvae or embryos to PAHs extracted from river sediment or cadmium ions resulted in heart deformities, impaired brain vascular development, pericardial oedema and reduced heart rate[104,105]. However, these cardiovascular toxic effects of PAHs and cadmium ions were attenuated by co-exposure to PS-NPs or PET-MPs. These antagonistic effects of co-exposure to particulate plastics may be attributed to reduced bioavailability and bioaccumulation of cadmium ions and PAHs.

Biphasic effects of PS-MP and cadmium ion co-exposure have also been reported in zebrafish embryos. Exposure to cadmium ions alone reduced the embryo heart rate, whereas the effect of co-exposure to cadmium ions and PS-MPs varied depending on the concentration of PS-MPs; lower concentrations of PS-MPs were associated with an increased heart rate, whereas higher concentrations of PS-MPs were associated with a decreased heart rate[105]. The embryo chorion may have a crucial role in the biphasic effects observed with PS-MPs; a high concentration of PS-MPs could damage the chorion membrane, facilitating the entry of cadmium ions into the embryo, whereas a lower concentration of PS-MPs could reduce cadmium ion bioavailability[106]. Further studies are needed to investigate the biphasic effects of PS-MPs and various contaminants in additional models and organisms.

Approaches to mitigating particulate plastic pollution

The extensive production and widespread use of plastic materials have inevitably led to the generation of plastic waste and particulate plastics. Mitigating or preventing the formation and associated risks of MPs and NPs remains a critical global challenge. Despite growing concern, few studies have specifically addressed the removal of MPs from environmental matrices[107]. Addressing particulate plastic pollution necessitates an integrated strategy, prioritizing both minimization of plastic production and enhancement of waste management infrastructure. The most sustainable and impactful approach is minimizing plastic production, which reduces the amount of material that can enter the environment[108]. Approaches to plastic degradation include oxidative photodegradation, thermal deterioration, biodegradation, catalytic decomposition, mechano-chemical degradation and ozone-induced degradation[109]. Although some methods, such as enzymatic degradation and advanced recycling processes, have been incorporated into some existing waste management frameworks, most of these approaches remain experimental.

Existing water treatment facilities employ processes and equipment capable of capturing conventional pollutants and a portion of MPs[110]. However, these systems were not designed explicitly for MP removal, and their efficiency largely depends on the type of treatment employed. The removal of microplastics therefore requires advanced solutions. Advanced oxidation processes that utilize reactive oxygen species, such as hydroxyl radicals, have shown promise for degrading MPs and microfibres (MFs). Nanomaterials such as TiO_2 and ZnO generate reactive oxygen species when exposed to UV light and thereby

promote oxidative degradation of plastic polymers, including polypropylene, PS, PVC, low-density polyethylene and high-density polyethylene, reducing the time required to decompose these plastics[111]. However, reactive oxygen species can only partially modify the surface chemistry and composition of MPs[112]. TiO_2 photocatalysis has achieved >90% mass loss for polyamide MFs, whereas traditional Fenton processes, which generate highly reactive hydroxyl radicals when H_2O_2 reacts with soluble iron (II) salts, achieve <30% oxidation of MP. Enhanced Fenton processes can degrade up to 96% of MPs under specific conditions (temperature >100 °C, reaction time of at least 5 h and pH ≤3)[113]. TiO_2 has been utilized in water treatment systems to degrade particulate plastics[114]. Surface coating of waste plastics with TiO_2 or ZnO could also potentially be used to accelerate degradation during waste management[115]. However, commercial adoption of these methods remains limited.

Natural biodegradation pathways struggle to break down particulate plastics, which are often fragmented into smaller particles rather than fully decomposed. Around 436 species of microorganisms have been reported to degrade plastics, including 66 different species of bacteria, with *Bacillus pumilus*, *Aspergillus fumigatus* and *Phanerochaete chrysosporium* being the most common[116]. Microbial enzymes such as cutinase, lipase and PETase, primarily hydrolases, catalyse the breakdown of polyester in the presence of water, reducing the molecular weight of the plastic monomer. In addition, a Gram-negative bacterium *Ideonella sakaiensis* 201-F6 has been demonstrated to degrade PET[117]. Pretreatment methods, such as heating, cooling and chemical processes, can enhance microbial attack by breaking polymers into monomeric units. Enzymatic degradation leads to the mineralization of these monomers, releasing CO_2, H_2O, CH_4, N_2 and other metabolic byproducts[118].

Substantial progress has also been made in the biotechnological approach to plastic recycling. A variant of leaf-branch compost cutinase, an enzyme initially identified in microbial communities from leaf-branch compost, has demonstrated high efficiency in breaking down PET, achieving 90% conversion on an industrial scale. A PET hydrolase, TurboPETase, has been engineered to degrade the remaining 10% of non-biodegradable PET. TurboPETase achieves almost complete depolymerization within 8 h at a solid loading of 200 g/kg. This advance creates new opportunities for polyester hydrolase engineering and polymer recycling initiatives but also holds promise in mitigating MP and NP pollution. TurboPETase and similar enzymes have the potential to be adapted for use in water-treatment facilities to break down PET-based MPs and NPs in wastewater streams or to be integrated into waste management systems to improve plastic recycling efficiency and reduce environmental plastic pollution[119].

Another approach to mitigating particulate plastic pollution is to use alternative materials to conventional plastics, such as bioplastics, glass, metal, paper, wood and textiles. In particular, conventional plastic packaging is increasingly substituted with materials that are perceived to be more environmentally sustainable[120]. However, such materials are not always more environmentally friendly than conventional plastic. For example, glass has a higher ecological footprint than lightweight plastics owing to its substantial weight, which increases energy demands during transportation. Consequently, implementing reuse strategies for plastics is critical to minimizing the overall environmental impact of packaging materials.

Conclusions

The ubiquitous prevalence of MPs and NPs in our daily lives, from the clothes we wear to the air we breathe and the food we eat, underscores the pressing need for further research and measures to address this escalating environmental and health concern. Numerous laboratories have conducted toxicological studies on particulate plastics using various cell and animal models. The results indicate that particulate plastics can cause oxidative stress and cytotoxicity, disrupt metabolism and impair immune function. Particulate plastics have also been shown to harm the kidney and cardiovascular system. In addition, exposure to particulate plastics can alter the composition of the gut microbiota and thereby not only contribute to gastrointestinal disorders but also have systemic effects, which could influence interorgan communication and contribute to disease pathogenesis. Precise quantification of human exposure to particulate plastics is challenging and further research is warranted to provide a deeper understanding of routes of human exposure, the resulting adverse effects and their impact on health outcomes. Such research is vital to inform environmental policy and public health strategies.

Improved degradation of particulate plastics via biotic and abiotic mechanisms could help to mitigate their accumulation and environmental contamination. Other potential approaches to mitigating exposure to particulate plastics include stricter regulations for plastic production, use and disposal, the promotion of safer alternatives to conventional plastics, enhanced environmental monitoring, improved water filtration processes and workplace protections. Public education approaches could also help to reduce the use of single-use plastics and encourage sustainable choices. Such actions are crucial to protecting ecosystems and human health.

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